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Blood Lead Levels in Children

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In their guest editorial published in *Environmental Health Perspectives*, Brown and Rhoads (2008) endorsed the Centers for Disease Control and Prevention's (CDC) position, maintaining the child blood lead level (BLL) of concern at the 10-μg/dL 1991 standard (CDC 2008). They added little substance to support the rationale.

Brown and Rhoads (2008) expressed surprise that the BLL–IQ relationship slope at low BLL is steeper than at higher BLL. Almost all studies examining BLL–IQ relationships have found this nonlinear form, as summarized in the pooled analysis of seven prospective lead studies (Lanphear et al. 2005). Nonlinear lead response is also found in child studies with math and vocabulary scores (Kordas et al. 2006) and fine motor and visual motor function (Wasserman et al. 2000), as well as many others. The authors doubt this relationship, wondering if "such a strong relationship is plausible, particularly as there are no directly relevant animal or *in vitro* studies that demonstrate" the relationship (Brown and Rhoads 2008). They are uninformed. To cite just a few relevant studies, lead inhibits δ-aminolevulinic acid dehydratase activity in humans (Murata et al. 2003) and inhibits peak current amplitude of acetylcholine-induced currents in cultured rat hippocampal neurons (Ishihara et al. 1995); in monkeys, increasing gestational lead resulted in increasing incomplete responses during acquisition of a fixed-ratio operant task (Newland et al. 1996), all with nonlinear dose response.

In their editorial, Brown and Rhoads (2008) hypothesized increased national IQ from decreased child BLL in the United States from the late 1970s to 2002 based on the nonlinear relationship. They claimed, without citation, that "there is no agreement that IQs have increased by 7 points." However, they ignored the Flynn effect (Flynn 1985)—the secular trend of IQ increase noted throughout the world. During the late 1970s–2002, the Wechsler Intelligence Scale for Children changed in content and standardization to account for the Flynn effect, making impossible long-term national tracking of IQ increase with this or any renormalized test. Although Brown and Rhoads (2008) cited no change in U.S. student reading scores, they failed to note that mathematics and science scores in the same longitudinal study increased significantly from 1982 to 1999 (Campbell et al. 2000).

Brown and Rhoads (2008) stated in their editorial that the CDC will not change the action limit because "no effective, feasible interventions to reduce BLLs in this range have been demonstrated." Maintenance of high BLLs in chronically exposed children after intervention emphasizes the need for primary prevention but does not address the issue of setting lower action targets.

Brown and Rhoads (2008) cited the CDC claim that "given current laboratory methods, risk for misclassification of children is high" for the current BLL of < 10 μg/dL. To be truthful, they should have changed "current laboratory methods to "current screening laboratory practice." The CDC-led National Health and Nutrition Survey study found the accuracy and reliability of BLL measurements < 5 μg/dL BLL adequate for describing national BLL (National Center for Health Statistics 2008). Potential misclassification with lower BLL limits is a policy issue, not a technical one.

The CDC maintains the old elevated BLL definition because they have found no effect threshold; that is, with no effect threshold, any new action limit would be arbitrarily defined. An effect threshold did not determine the 1991 CDC action limit. Evidence in 1991 suggested that 10 μg/dL would protect most children from lead effects (CDC 2005). But it was wrong. Evidence today points to risk for developmental damage down to the lowest BLLs explored in prospective studies, effectively 1 μg/dL (Lanphear et al. 2005).

Brown and Rhoads (2008) reserve the label "lead poisoning" to BLLs > 10 μg/dL. They propose an action plan on a primary prevention scale not yet present, requiring years of legislative and bureaucratic wrangling for enaction and implementation. The proposed interagency partnerships will focus on "housing where children have repeatedly been identified as having elevated BLLs," without changing the definition of elevated BLLs to expand the focus below the current criterion.

Although there are strong reasons to promote primary prevention to protect children from lead, Brown and Rhoads (2008) use disingenuous opinion favoring an incomplete, flawed plan that guarantees long delays. Waiting years to implement a new primary prevention plan and neglecting the majority of exposed children is indefensible. The CDC must redefine the standard, providing a benchmark by which to judge further progress and redirecting the focus to all those affected. At present, scientific evidence supports revising that standard to well below 5 μg/dL. How unfortunate for our children that political will supports only more delay and denial.

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Stephen J. Rothenberg
Centro de Investigación y de Estudios Avanzados Instituto Politécnico Nacional
Mérida, Yucatán, México
E-mail: drlead@prodigy.net.mx

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Blood Lead Levels: Rhoads and Brown Respond

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Rothenberg states that the level of concern for blood lead in children should be lowered from 10 μg/dL, but he provides little cogent rationale for doing so. As basic science support, he

cites studies that *a*) exposed isolated neurons to a 3 μM solution of lead (Ishihara et al. 1995), a concentration that is 5,000 times the plasma levels expected in a child with a blood lead level (BLL) of 5 μg/dL (Manton et al. 2001); *b*) a study of squirrel monkeys exposed *in utero* to maternal BLLs in the 20- to 70-μg/dL range (Newland et al. 1996); and *c*) a study of occupationally exposed workers with a median BLL of 17.1 μg/dL that contains no data on neurologic effects of lead (Murata et al. 2003). Rothenberg's choice of these citations emphasizes how little basic science work has been done on neurodevelopmental effects at the very low levels of lead under discussion.

There was a 90% decrease in U.S. childhood BLLs from the late 1970s to the late 1990s (Pirkle et al. 1994). If the regression coefficients relating BLLs < 10 μg/dL to cognitive functions are taken at face value, they predict a population-wide, half standard deviation of cognitive improvement as a result of this fall in blood lead—a remarkable shift that should have substantially increased the number of very bright students with IQ > 135. To our knowledge, no such effect has been noted in the education literature, nor is it evidenced, for instance, among the increasing proportion of U.S. students admitted to U.S. graduate programs (Basken 2006).

What measures can be used to look more formally for this IQ improvement? IQ itself is problematic because the Flynn effect and adjustments in test instruments make secular changes in IQ hard to interpret. The teaching content in science and math has likely shifted over this period. Therefore, in our editorial (Brown and Rhoads 2008) we cited reading scores that measure a key skill that has been identified repeatedly as being affected by lead exposure among other factors. Campbell et al. (2000) reported that reading scores, examined with a suitable time lag in large nationally representative samples of children, were virtually unchanged over the critical period of declining lead levels. Rothenberg is correct that modest gains in math and science were recorded, but these changes could easily have other explanations, and they do nothing to explain the absence of any signal in reading scores. Although there may be other explanations for this absence, the simplest explanation of this paradox is that the published regression coefficients relating BLLs < 10 μg/dL to cognitive measures, all of which come from observational studies, are biased. This possibility is suggested by the steepening of the IQ curve at low lead levels [Centers for Disease Control and Prevention (CDC) 2005b].

Regardless of one's view of the above evidence, it is important to recognize that virtually all of the progress made in eliminating childhood lead poisoning has been through primary prevention—the control or elimination of lead sources before children are exposed. This approach has lowered the proportion of 1- to 5-year-old children with BLLs > 10 μg/dL from well above 50% 30 years ago to 1.6% in 1999–2002 (CDC 2005a). The percentage is almost certainly lower today. Primary prevention has been proven to work and deserves the continuing attention that we described in our editorial (Brown and Rhoads 2008). Primary prevention can, and should, include increased attention to controlling exposures from lead paint hazards, imported foods, medicines, cosmetics, and toys. Renewed emphasis on screening with a lower BLL of concern would be expensive, intrusive to families, and hard to justify in the absence of proven, practical strategies for reducing lead levels in identified children. Further, it would likely deflect needed resources away from the primary prevention effort.

The CDC, in collaboration with federal, state, and local agencies, has outlined and begun to implement a comprehensive, society-wide effort to prevent lead exposure in children while maintaining efforts to identify and treat children with elevated BLLs (CDC 2005b). The CDC has also developed specific recommendations for health care and social service providers, scientists, and public health practitioners who are interested in actively participating in these primary prevention efforts by providing valuable leadership and expertise (Binns et al. 2007; CDC 2005b). By working together with federal, state, and local agencies to foster expansion of primary prevention services, these child advocates can accelerate achieving our mutual goal—lead-safe environments for the nation's children.

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George Rhoads
Advisory Committee on Childhood Lead Poisoning Prevention
School of Public Health
University of Medicine and Dentistry of New Jersey
Piscataway, New Jersey

Mary Jean Brown
Lead Poisoning Prevention Branch
Centers for Disease Control and Prevention
Atlanta, Georgia
E-mail: mjb5@cdc.gov

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